In several cases he had advised the counsel to settle the case, and recovery had occurred in a few months.

It was a curious fact that patients suffering from organic injury usually did not have neurasthenia. The exaggerated manner of the neurasthenic contrasted curiously with the calmness of the man with serious organic injury.

The psychical results of injury would entitle the sufferer to some recompense, but of course not to so much as miliary hemorrhages or other wide-spread disease.

Dr. ZENNER referred to the fact of hysterical symptoms conjoined with organic disease, referring to a case of lead poisoning associated with tremor, which disappeared under hypnotism and after two or three applications was cured. Deception on the part of the patient should not, on the other hand, lead the exclusion of other serious disease. Pure malignering was a very rare occurrence. Deception n itself presupposed disease.

Dr. KNAPP recognized the tendency to auto-suggestion and the exaggeration of symptoms produced by these medico-legal trials, but neither sources of error were present in his cases, as suits for damages were not in progress, and all had been anxious to get well.

The following paper was read:

THE CORTICAL LOCALIZATION OF THE CUTANEOUS SENSATIONS.

By CHARLES L. DANA, A.M., M.D., New York.

I desire to present the evidence, so far as it is now attainable, regarding the question of the cortical localization of the cutaneous senses, viz., tactile, thermic and pathic.

It is well known there are now two very antagonistic views with regard to this subject. Ferrier contends that these centres are in the hippocampal gyrus and gyrus fornicatus. He bases this view upon experiments upon eleven monkeys, certain anatomical considerations, and the clinical fact that very often lesions of the convex motor cortex of the brain do not cause any sensory symptoms. His experiments are confirmed by those of Horsley and Schäfer. No positive pathological evidence is brought forward to support this view.

The other view is that the cutaneous sensory centres are more or less identified with the motor centres, being situated perhaps a little more posteriorly. Munk, Tripier, and Luciani and Sepilli, in particular, contradict the statements and experimental evidence of Ferrier. Luciani and Sepilli record experimental observations made upon ten dogs and four monkeys, besides pathological observations in forty-seven cases.

There is still a third view, viz., that both the motor cortex and the limbic lobe are concerned in the representation of cutaneous sensations.

Thus, in brief, the matter stands at present.

In pursuing an investigation of this kind, we make use of several kinds of evidence:

1. That obtained by clinical and pathological observation on man.
2. That obtained by experiment upon the lower animals.
3. That obtained by studying the development of special tracts in the embryo.
4. That obtained by the study of brains genetically imperfect.
5. That obtained by studies in comparative anatomy and physiology.

The atrophy method of Gudden cannot be directly applied to this question. Of the foregoing, it is the first class of evidence which is by far the most important and decisive, outweighing all the others. Perhaps the next most important is the experimental evidence obtained by observations on monkeys. At least it seems to be shown that the representations of motor areas in the brain of the *macacus* is closely identical with that in the brain of man.

My paper deals chiefly with pathological and clinical data, and these I shall present first.

I. A few cases of lesions of the brain cortex with sensibility disturbances were collected by Nothnagel in his work published in 1879. They were insufficient for him to form a conclusion as to localization. Exner in 1880 collected twenty-two cases, Starr in 1884 collected forty-one cases, Luciani and Sepilli in 1885 forty-seven cases, illustrating sensory localization, these latter including some of Starr's and Exner's. I have consulted the originals in Starr's list, and eliminated some in which the reports did not seem satisfactorily complete. Adding the remainder to Luciani and Sepilli, I found that I had eighty cases. Searching through the literature of the last four years, I have been able to find fifty-eight cases. To these I add four cases of my own, making a total of one hundred and forth-two.

These are all cases in which symptoms were observed during life, and autopsies were made, except in nine instances. In these latter there was injury of the skull and rupture of the middle meningeal artery. Trephining proved the nature of the lesion. Beside this, in the way of negative evidence, I have collected twenty cases of lesions of the gyrus fornicatus or hippocampus.

The cases have been tabulated in such a way as to show the location and nature of the lesion, the leading symptoms aside from the anæsthesia, and the kind and degree of the disturbance.

The numbers of the cases have then been marked upon charts of the brain cortex somewhat after the manner of Exner and Naunym; only, instead of dividing the brain surface into small squares, as was done by the observers mentioned, I have divided the chief convolutions into thirds and halves. In reading descriptions of post-mortem appearances, it seems to have become a custom to describe lesions as occupying certain thirds, as, for example, of the frontal, central, and temporal convolutions. Descriptions are rarely more minute than this. In reading over some of Exner's cases, one is a little astonished at the confidence with which he marks down a lesion in a minute square from the vaguest possible description. A good deal of scientific imagination must be used in dealing with the squares. The divisions I have made therefore are, I venture to think, more natural, less arbitrary, and better fit into ordinary descriptions of post-mortem findings. The results of my tabulation upon the brain charts are shown here. *Figs. 1 and 2.*

These charts, however, do not give a perfectly just idea of the sensory areas, for I have also marked down the areas involved in extensive or multiple lesions, for example, lesions involving both central and occipital convolutions. In these cases the lesion in the extra motor zones caused other symptoms, or, as shown by other cases, had nothing to do with the cutaneous sensory disturbances. It will be found that no lesion in the occipital, temporal, or anterior part of the frontal lobes caused cutaneous sensory troubles unless there was also some involvement of the motor areas. Even in the inferior parietal lobule there are but four lesions causing cutaneous sensory disturbances, and not extending into the motor regions.

The clinical and pathological evidence thus collected shows that the motor areas of the cortex contain also the representation of cutaneous sensations.

A study of the cases shows that the sensory centres for special parts of the body, i. e., face, arm, or leg, are in general identical with the motor centres for those parts, but are larger and more diffuse. It takes an extensive and, as it seems to me, a deep lesion to cause marked anæsthesia. For example, in the cases in which there was distinct and very marked anæsthesia, the pathological condition was:

Abscess, - - - - - - - -	2
Softening (from thrombosis or encephalitis),	27
Tumors, - - - - - - - -	7
Meningitis, - - - - - - - -	3

In very few cases does one find a record of hæmorrhage; hence, the sudden appearance of anæsthesia with hemiplegia is rather a sign of necrotic or inflammatory softening than of rupture of vessels. Surface clots, however, do sometimes cause incomplete hemianæsthesia along with the paralysis.[1]

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[1] Thus Wiesmann, in a collection of over two hundred cases of fracture of the skull with rupture of the middle meningeal, found incomplete hemianæsthesia in seven cases. I have noted two other cases, one of my own and one of Wilkes's.

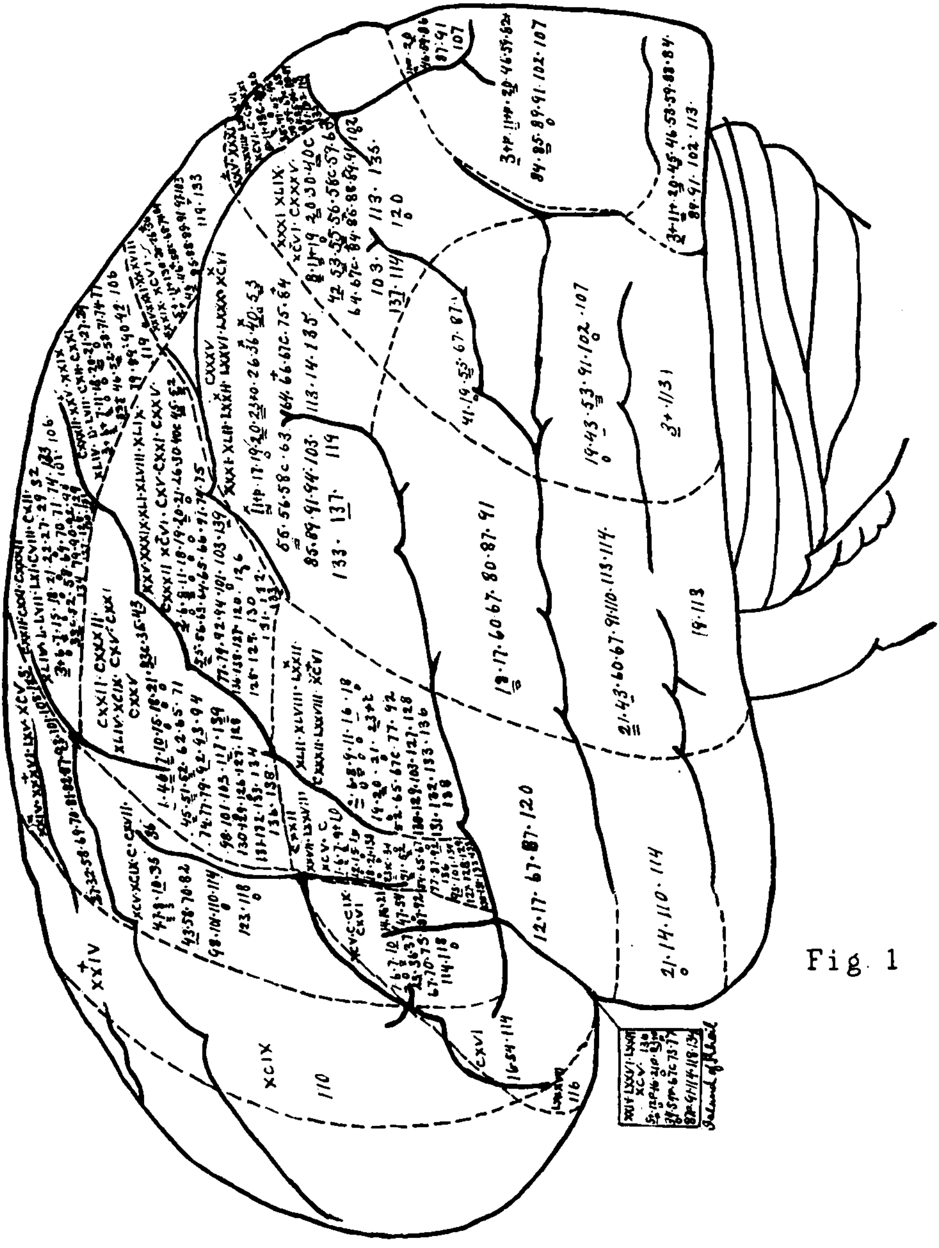
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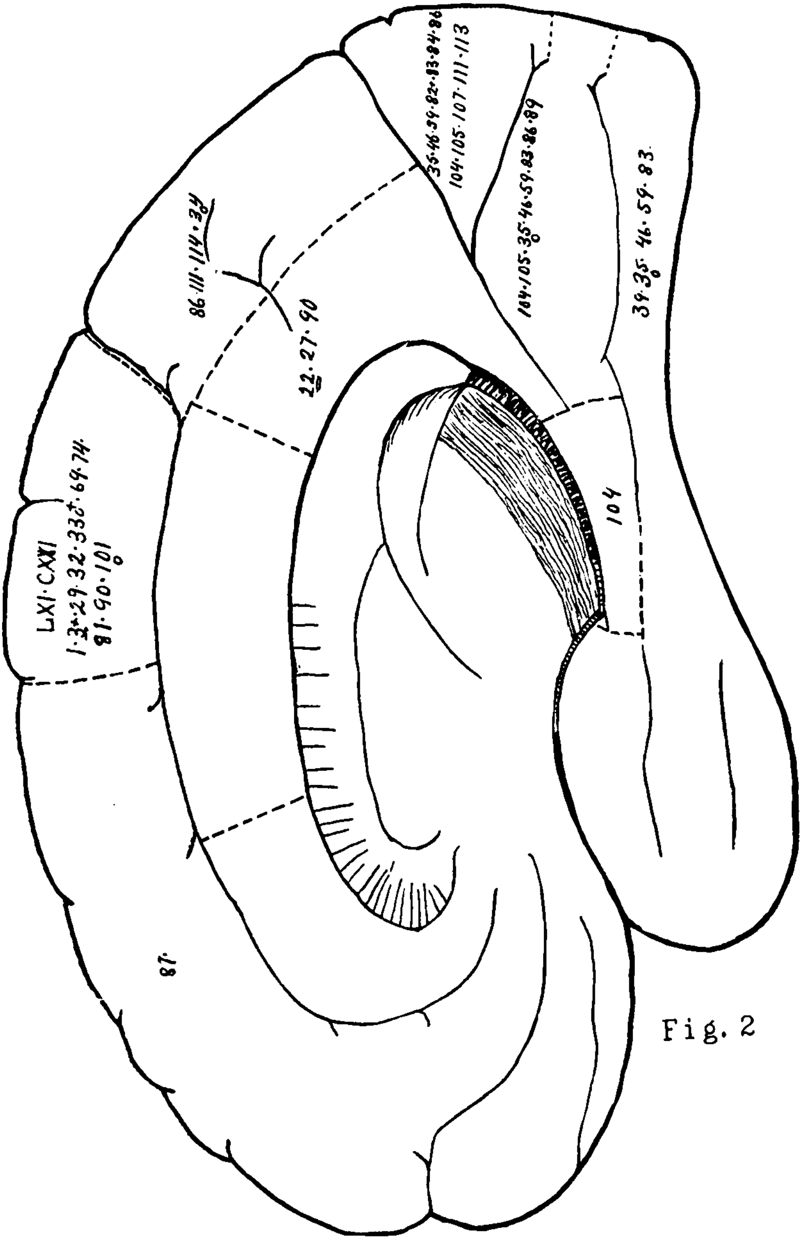
Fig. 1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81

Fig. 2

In the cases of paræsthesia alone, the lesions were:

Tumors,	16
Softening,	7
Meningo-encephalitis,	5
Meningitis,	1
Clot,	3

Thus it seems that the slowly developing lesions oftener produce only paræsthesia.

An analysis of the cases with reference to localization in the right or left hemisphere shows a preponderance of nearly two to one in favor of the left hemisphere, especially in the central convolutions, e. g.:

Frontal lobes, except paracentral lobule—	
Right hemisphere,	14
Left "	27
Not stated,	1
Ascending central—	
Right,	45
Left,	85
Not stated,	4
Superior parietal—	
Right,	17
Left,	12
Not stated,	4
Inferior parietal—	
Right,	17
Left,	25
Not stated,	3
Paracentral—	
Right,	4
Left,	6
Not stated,	2
Island of Reil—	
Right,	12
Left,	5
Not stated,	2

In the island of Reil the lesions are much oftener on the right side.

The preponderance indicated may be due to a greater frequency of recorded lesions in the left hemisphere. The paræsthesiæ alone were caused by lesions about equally distributed in each hemisphere—r. 13, l. 12. The severer cases of anæsthesia were also due to lesions about equally distributed—r. 17, l. 14.

It may be stated here that the cases collected, so far as they go, confirm the view that the seat of muscular memories is in the inferior parietal lobule.

An analysis of the different cases of paræsthesia alone, of analgesia, and of tactile, thermic, and pathic anæsthesia, two or all forms being present in the cases, is shown on the accompanying charts.

FIG. A.

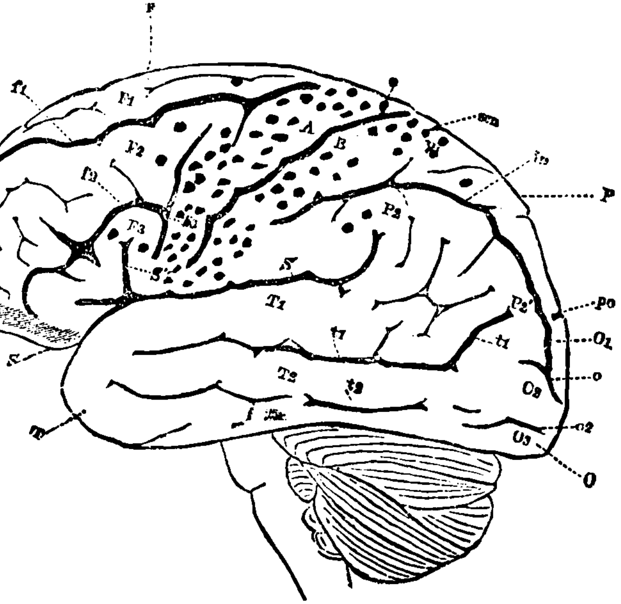
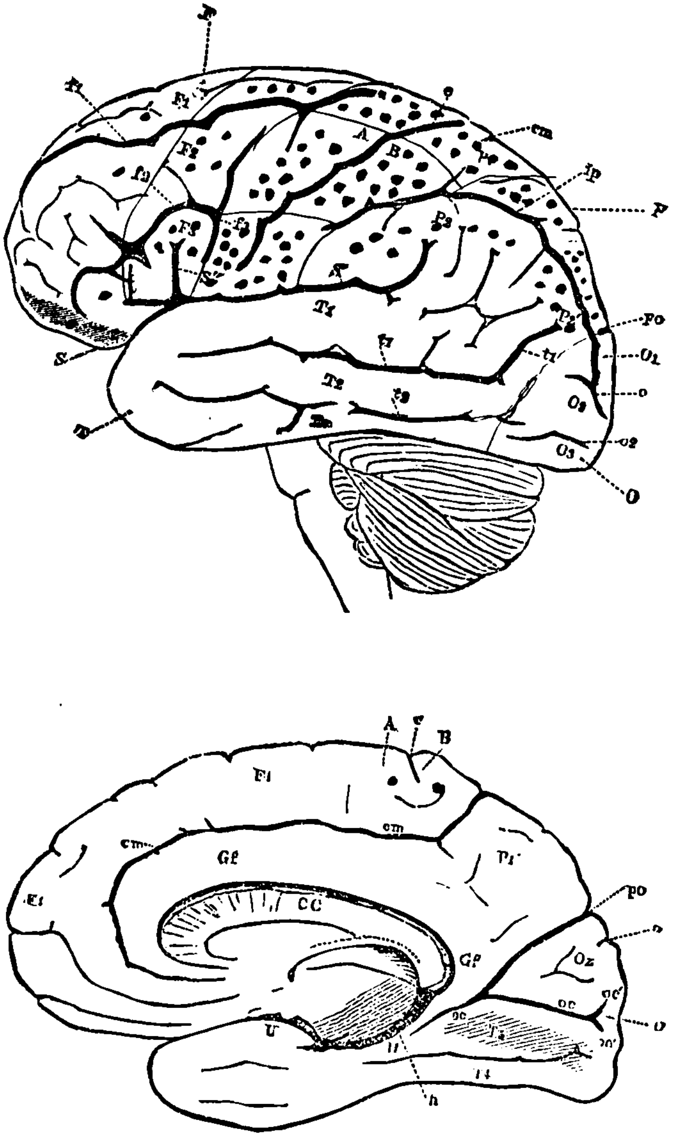


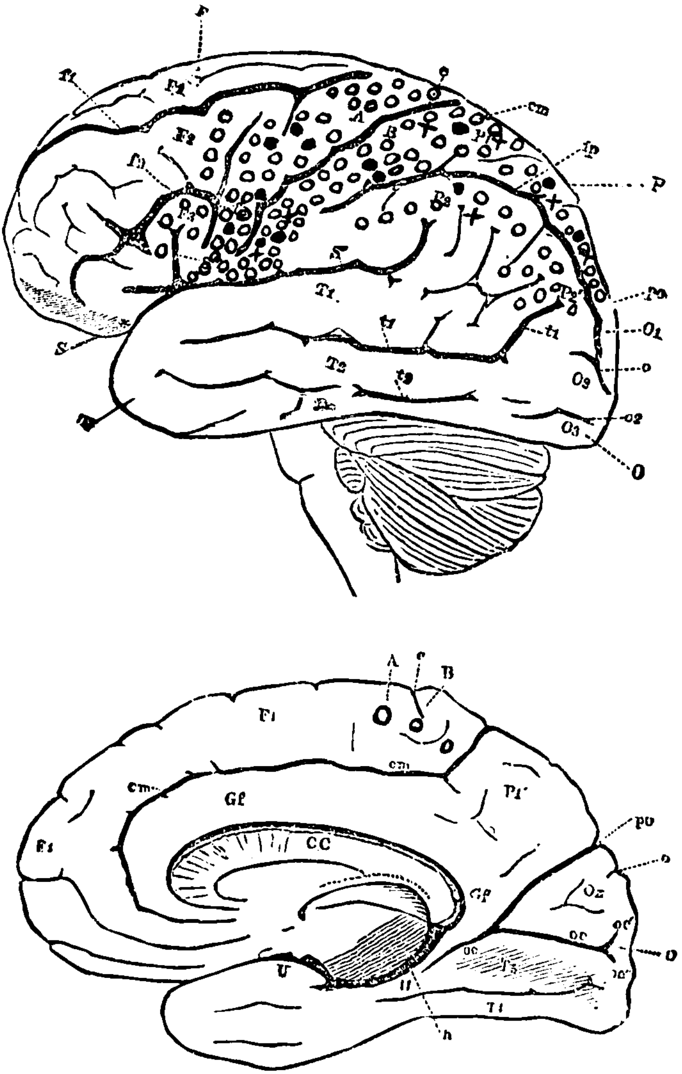
Diagram showing localization and number of lesions in cases in which there was analgesia.

Figs. B, B1.



Diagrams showing localization and number of lesions in which there was paræsthesia.

FIGS. C, C'.



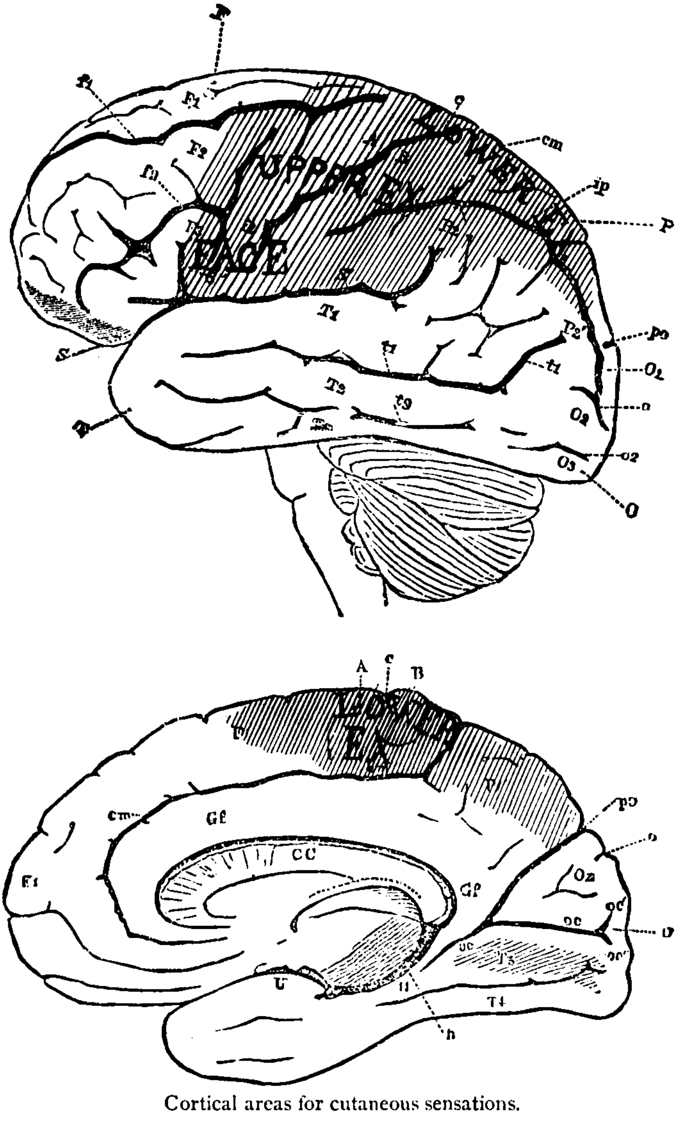
Diagrams showing number and localization of lesions :

● = tactile anæsthesia.

+ = tactile and thermic anæsthesia.

○ = combined tactile, thermic, and pathic anæsthesia, or tactile and pathic anæsthesia alone.

FIGS. D, D¹.



Cortical areas for cutaneous sensations.

The tables and cuts do not show any especial difference in the localization of the three different sensations. If anything, tactile sensation seems to be more strictly confined to the motor areas. On the whole, the pathological histories show that tumors and slowly growing lesions in the motor areas, either in or just below the gray matter, produce paræsthesia and slighter degrees of anæsthesia. Softening, thrombotic or inflammatory, of the same areas coming on suddenly produces localized anæsthesia if it is extensive, and completely destroys the parts. Pressure clots from meningeal hemorrhage, if extensive and severe, will cause partial anæsthesia of the opposite side, as well as profound hemiplegia.

There is no known region of the cortex, lesion of which will cause anæsthesia without accompanying hemiplegia. The nearest approach to this is in lesions of the supramarginal lobule, where a slight degree of tactile anæsthesia without paralysis has been occasionally noted.

It is not possible for a cortical lesion to cause total unilateral anæsthesia without the lesion being so extensive as to be rapidly fatal. That there is a total crossing of the cutaneous sensory nerves, however, seems probable from the fact that the crossing is practically total in the cord, medulla, pons, and capsule.

Besides, lesions of the lower part of the central gyri have produced almost absolute anæsthesia as in Case I. of my own and Case III. of Petrina. It seems like an "absolute" cortical area for sensation.

From the foregoing we deduce further general laws, that *cutaneous anæsthesia of organic cortical origin is always limited to or more pronounced in certain parts, e. g., the face, or arm, or lower limb of the body, and it is generally incomplete.*

Total hemianæsthesia is either of functional or subcortical origin.

Cortical anæsthesia is always accompanied with some degree of paralysis.

II. *The experimental argument.*—It must be admitted that at present many facts obtained by experiments upon monkeys favor the idea that the limbic lobe is the seat of cutaneous sensations. We must remember, however, that Ferrier is alone in denying that anæsthesia is caused by removal of parts of the motor areas. Schiff, Luciani,[1] Tripier, Munk, all have noted sensibility disturbances after these lesions. The positive experiments made upon the limbic lobe are as yet few in number, and the operation is confessedly a very difficult one.

III. *The embryological evidence.*—The study of the development of sensory tracts in the embryo has so far failed to show that any but a very small part of the sensory tract turns in toward the limbic lobe. Most of the fibres pass up in the direction of the parietal lobe (including the postcentral convolution). Von Monakow has shown, by the atrophy method, a connection between the parietal cortex and a part of the sensory tract.

IV. *The evidence from teratology.*—Zuckerkandl[2] has collected two cases in which infants were born with absence of the olfactory lobe on one or both sides. In these cases there was decided lack of development of the cornu Ammonis; the middle and posterior horn of the lateral ventricle in one case was absent, and the hippocampus and gyrus fornicatus in both cases were small.

He refers to two other cases shown him by Kundrat, but I cannot find any details.[3]

V. *The anatomical and comparative anatomical evidence.*—Mr. Victor Horsley finds in the anatomy of the cortex an evidence of sensory function. At the discussion on cerebral localization before the Congress of American Physicians and Surgeons, September 19th, 1888, he said that, as the large pyramidal cells were undoubtedly motor, the smaller cells were probably sensory, and he showed the following diagram:

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[1] Luciani and Sepilli, in ten dogs and four monkeys, found that extirpation of motor region caused an. and atax. Tactile sense much affected, muscular sense even more, pain and thermic sense less.

[2] Ueber das Riechencentre.

[3] Tiedman reported the case of a child born without olfactory nerves, Ammon's horn and fornix not developed perfectly; cited by Zuckerkandl, p. 107, op. cit.

Rudolphi, 1814, cited by Zuckerkandl, p. 107, op. cit., described the brain of child with absence on right side of I., II., III., IV., VI., cranial nerves. The corpus callosum was shorter on right side, the right lateral ventricle smaller, anterior horn short, descending and posterior absent, Ammon's horn very small, ditto fornix and corpora albicantia.

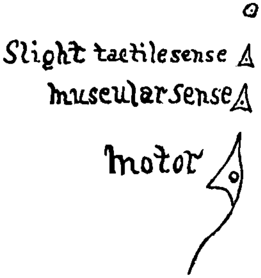


Diagram A.

Muscular sense, as I understand him, he takes to be compounded of (*a*) a sense of present movement or innervation, (*b*) a memory of past movement. It can only be the first, as it seems to me, which is in the motor areas.

The muscular memories, by which co-ordinated purposive movements result, seems to be, so far as is shown by the evidence of human pathology, in the inferior parietal lobule.

Finally, the evidence of comparative anatomy is quite against the view that the limbic lobe is the centre for tactile or pathic sensations.

The elaborate comparative anatomical studies of Broca some years ago and of Zuckerkandl (Ueber das Riechencentrum, F.Enke,Stuttgart, 1887) show that in anosmic animals like the dolphin this lobe is rudimentary. In animals with a highly developed olfactory nerve like the dog, this lobe is very large. Zuckerkandl gives the following table showing the relative development of the gyrus fornicatus:

GYRUS FORNICATUS.

ABSENT.	RUDIMENTARY.	PRESENT.
Monotemes.	Cetacea.	Ungulates.
Marsupials.	Primalis.	Carnivora.
Some families of chiroptera.	Some prosimians.	Insectivora.
		Man.

And Zuckerkandl concludes that the limbic lobe is the seat of olfactory sensation.

Objections.—In answer to the facts which I have brought forward, two objections will be raised: first, the large number of negative cases in which no sensory disturbance occurred despite extensive destruction of the motor tract; second, the positive experiments of Ferrier and of Horsley and Schäfer upon the limbic lobe in monkeys. To the first of these objections one cannot yet offer a perfectly satisfactory answer. To be sure, it is not true, as Ferrier states, that one negative case upsets the whole argument. This is not logic, or, if it is, it is a logic which works both ways. We would have to give up our cortical motor centres; for there still occur every now and then cases in which there is profound and extensive destruction of the motor areas without paralysis, as witness the cases recently reported by Byrom, Bramwell (*Intracranial Tumors*), and Gaskill (*Brit. Med. Jour.*, vol. I., 1888). On the other hand, in late years, with more careful observation, the cases of serious lesions of the motor areas without sensory disturbance are becoming more and more rare. Incomplete observation, late observation when compensation has occurred, small lesions, slowly developing lesions, are the factors which may explain the frequent absence of sensory disturbances.

As to the second point, a word may be said first regarding Ferrier's anatomical argument, viz. (loc. cit., p. 326), that there is a bundle of fibres in the outer third of the foot of the crus which does not degenerate downwards, which is connected probably with the sensory tract, and which bends round towards the hippocampus.

This statement has been disproved by the observations of Bechterew, who shows that this bundle does degenerate downward, and that it connects the frontal lobes with the pons nuclei fibres, passing thence to the contralateral cerebellum (*Archiv f. Psych.*, 1888). The experimental facts adduced by Ferrier are, however, most weighty. I am not prepared to deny that it is beyond possibility for the limbic lobe to be shown to be a centre for cutaneous sensations in man; but the facts of human pathology so far do not give it the slightest support. The few cases of lesion of the gyrus fornicatus collected by Exner showed it to be a latent region. I have been able to find other cases, making in all twenty, in which the gyrus fornicatus or hippocampus was more or less involved. In none of these was any anæsthesia that could be fairly attributed to the lesion in the gyr. fornicatus observed.

LESIONS OF GYRUS FORNICATUS AND HIPPOCAMPUS.

1. Von Monakow, Arch. f. Psych., xi. Sarcoma of falx pressing on gyr. forn. No symptoms.
2. Von Monakow, Arch. f. Psysh., xiv., Hft. 3, 1883. Quoted by Luciani and Sepilli. Softening of lingual lobe and posterior part of hippocampus, 1st and 2d temp. cuneus. Blindness, hemiplegia, dementia.
3. A. McL. Hamilton, N. Y. Med. Jour., 1882, p. 575. Uncinate slightly, also hippocampus; sensory epilepsy.
4. Wildebrandt, Arch. f. Ophthal., 31, p. 119. Cuneus involving hippocampus and lingual (5th T.) slightly. Hemianopsia, slight hemianæsthesia.
5. J. A. Vorthnis, Brain, July, 1886. Upper 3d, central convol. Paracentral lobule extending into g. fornicatus. Hemiplegia and, later, hemianæsthesia in foot.
6. Mills, Phila. Med. Times, Jan. 18th, 1879. Tumor of right posterior ½, 1st of and 2d frontal, small segment of anterior gyrus forn. and corp. callosum. Spasm and mental disturbance.
7. Seguin. Tumor and cyst of paracentral lobule, part of first frontal, and involving part of gyrus fornicatus. No marked anæsthesia at any time. Jacksonian epilepsy.
8. Bristow, Brain, 1884, Oct., Case 2. Tumor corp. callosum involving 1st and 2 frontal postcentral and anterior ½ corp. callosum. No anæsthesia.
9. Reinhard, Arch. f. Psych., xviii., Case 13. Softening, fusiform, cuneus, convex occip. con., sup. parietal convolutions, ang. gyr., 2d temp. con. Blindness, motor aphasia, dementia.
10. Seguin, Arch. de Neurolog., 1886, No. 32, Case 45. Softening of right cuneus, 5th temporal, part of 4th, extending forward into part of hippocampus. Hemianopsia, slight ataxia.

11. Haab, 1882, quoted by Seguin, loc. cit., Case 28. Softening lower ½ cuneus, part of hippocampus and 5th temp. conv., extending into ventricle. Hemianopsia, temporary hemiplegia, no hemianæsthesia.
12. Huguenin, quoted by Seguin, Case 29, loc. cit. Tumor of right cuneus, extending forward and involving slightly the hippocampus. Hemianopsia.
13. Cassy, quoted by Luciani and Sepilli, op. cit., p. 324. Complete atrophy of cornu Ammonis and hippocampal gyrus. No anæsthesia. See also cases of atrophy of this region in epilepsy cited by Meynert, Snell, Hencks, Pfleger, Tamburini, etc.
14. Only general convulsive attacks. Involvement of first frontal præcent., and anterior ½ of gyr. forn. Charcot and Pitres (Rev. Mensuelle, 1878, p. 810, obs. xix).
15. No motor or sensory disturbance. Involvement of occipital convexity, cuneus, præcuneus, and posterior of gyr. forn. Boyer (Les. Cortic, p. 58, obs. 31).
16. General paralysis; no anæsthesia or paraly sis. Lesion of frontal lobes and anterior 3d of gyr. forn. Baraduc (Bull. de la Soc. Anat., Marz, 1876, p. 277).
17. Tumor involving inferior and anterior surface of temp.-sphen. lobe and extending into hippocampus. Only slight visual disturbance; right hemiplegia. Boyer (Les. Cortic., p. 48, obs. ix).
18. Claus, Der Irrenfreund, 1883, No. 6. L.—Softening (1) posterior ½ fusiform lobe or T.; (2) spot of softening 3¼ ctm. long in frontal lobe, 5 ctm. behind anterior end gyrus fornicatus, in its white substance close under the cortex. Mental disturbances, aphasia, no paralysis or anæsthesia.
19. Bannister. This was a case reported to me verbally by Dr. Bannister. The patient was a lunatic who died with general cerebral symptoms, but without any general anasthesia. A very finely limited, cyst-like cavity was found occupying the lower part of the paracentral lobule and the adjacent part of the gyrus fornicatus. It was about 1½ inches in diameter.
20. M. Jastrowitz. Localization un Gehirn, Leipsig and Berlin, 1888, p. 49, Case VI. Tumor of right upper central convolutions, and lower anterior part of præcuneus and adjacent gyrus fornicatus. Hemiplegia of leg and arm with some loss of cutaneous sensation which, however, followed the gradual deveelopment of the paralysis.

Again, anæsthesia is not a symptom of tumors of the corpus callosum.

As to the theory that cutaneous sensations may be represented in both the motor areas and the limbic lobe, the case cited above by Jastrowitz (Case XX.) is instructive. Here there was a tumor with softening in the central convolutions and another tumor destroying part of the gyrus fornicatus. Yet the anæsthesia was not marked and it progressed with the paralysis. There was. however, decided muscular anæsthesia.

It is hard to conceive of a sensory mechanism so complicated and clumsy as that required by a hypothesis of compound sensory centres, one in the limbic lobe and one in the motor cortex; and no one has, I think, ventured to explain how much an anatomical arrangement could be put in harmony with the psychology of sensation. The mean reaction-time for tactile sensation is a little longer than that for sound, a little shorter than that for sight, (Wundt), e. g.

Sound,	0.167 sec.	or about	1-6 sec.
Touch,	0.213 "	"	1-5 "
Sighi,	0.222 "	"	1-5 "

The mean figures given by Hirsch, Hankel, Exner, Auerbach and Kries give the same relations. If the tactile senses were a compound mechanism involving the interaction of two centres, a longer reaction-time might be expected. As it is, since tactile sensations have a longer distance to travel on the peripheral nerves it may be fairly inferred that the actual cerebral portion of the tactile sensation reaction is shorter than that of sound or sight-sensation.

The objection which may be raised against a number of the cases quoted, that the lesion was not a purely cortical one, and that by pressure or extension, or involvement of the posterior capsular fibres anæsthesia was produced will not hold for a very large number of cases.

Owing to the immense typographical difficulties of printing an elaborate tabulated analysis of 134 cases I have omitted publishing it. I am certain, however, that *no amount of scrutiny can explain away the numerous cases in* *which superficial cortical lesions have caused monoplegias and monoanæsthesias.* I would refer as good examples of cases that illustrate anæsthesia from purely cortical lesions to Cases III. and VI. of Petrina, *Ztschr. f. Heilk, 1881*, Case I. of Luciani and Sepilli, Loc. cit. p. 302; case of Leyden, Beitrag. zur Lehre von der Loc. im Gehirn, 1888, p. 13.

Some of Reinhardt's cases (loc. cit.) also furnish examples of superficial lesions causing anæsthesia and paralysis.

Finally, it has been plentifully shown that the anterior part of the frontal lobes, the occipital and the tempero-sphenoidal lobes, are latent regions so far as cutaneous sensory disturbances are concerned.

If now we can exclude the gyrus fornicatus and hippocampus, as I believe we can, nothing is left as centres for these sensations but the central and the parietal lobes. My cases certainly appear to show that it is not in the parietal, lobes, if we except the upper lobule and post-central gyrus which are motor areas. By exclusion, then, as well as by positive evidence, we must conclude that the sensory and the motor areas are identical.

Conlcusions.—The cortical areas for touch, pain, and probably temperature, are identical with the motor areas.

The representations for the different parts of the cutaneous surface are, in the main, identical with the corresponding motor segments.

The areas for the different segments overlap and diffuse into each other.

The brain cortex of the left hemisphere, except the island of Reil, shows lesions causing sensibility disturbances twice as often as the right.

The anæsthesia produced by lesions in these areas is partial, as a rule; it may be total for a limited area, but never total for the entire half of the body. The anæsthesia from cortical lesion is limited to or more pronounced in certain parts of the opposite side, such as the face, arm, or leg.

Slowly developing lesions like tumors are especially apt to cause paræsthesia or sensory auræ if there is epilepsy. Sudden or extensive softening causes the most marked and profound forms of anæsthesia. This softening need not necessarily be deep but is often so.

Pressure lesions like superficial clots can cause some anæsthesia, but always with pronounced hemiplegia. Superficial syphilitic and tubercular lesions may cause partial anæsthesia.

No case of pure lesion of the limbic lobe has yet been observed (and reported) in which anæsthesia was produced. This lobe, or a part of it, is probably the seat of olfactory sensation.

The comparatively slight involvement of sensibility in lesions of the motor cortex has yet to be satisfactorily explained. It may be that each body segment has a larger area of representation on the cortex for sensation than for motion; or it may be that each segment is represented to some extent in both hemispheres, and hence compensation easily occurs. It is just possible that there is some sensory function in the limbic lobe.

The occurrence of slight sensibility disturbances with spasms and paralysis, points to lesions high up near or in the cortex.

Profound hemianæsthesia, with or without motor symptoms, points to a lesion lower down in the internal capsule or still nearer the cord.

Hemianæsthesia is always accompanied with some spasmodic or paralytic symptoms.

APPENDIX.

The following is a record of my own cases:

CASE *I.—Sudden right hemiplegia, motor aphasia, dysphagia, right hemianæsthesia very marked in face, intelligence dulled but present, early rigidity, hyperæsthesia on right side. Progressive weakness, bed-sores. Death in two months. Autopsy. Yellow softening; deep involvement of 2d and 3d left frontal and part of lower half of precentral convolutions, including cortex.*

A. G., female, age 70, native of Ireland, widow. No family or personal history obtained, except that the patient was quite well up to April 21st. On the morning of that day she suddenly became unconscious, and fell to the floor.

She was found to be paralyzed on the right side. She could not swallow. Her condition remained unchanged until admission next day.

On admission she was still but partially conscious. The right side of the face and right arm were partially paralyzed. There was some paralysis also in the leg. There was marked rigidity in the arm and leg. Sensation, motion, and reflexes were normal on the left side. *Sensation, tactile and pathic, was nearly or quite abolished on the right side of the face.* The mouth was drawn to the left side, and the right corner of the mouth was lower than the left. The right cheek was more flaccid than the left, and the right eye was not so closely shut as the other; it was suffused and congested.

Patient does not speak, but comprehends some remarks addressed to her. Her intellectual power is, however, notably impaired. She grasps anything with her left hand when told to do so, and, when asked to put out her tongue, attempts to pull it out with that hand. She continually puts her hand to the left side of her head, as if in pain. She cannot swallow, and grows very red in the face on making the attempt. Her eyes are turned to the left.

Patient frequently yawned. Left axilla more moist than the right.

Pulse slow, resp. normal, temp. 100.

Urine clear, normal in appearance, s. g. 1030, and contained no albumen.

Patient is a large and muscular woman. Her nutrition is good; her liver and spleen are normal. The signs of general bronchitis are present.

Ordered ol. tiglei gtt. ij. in the afternoon. During evening she had two passages.

Of milk and whiskey ℥ij. were introduced into patient's stomach by means of the stomach pump. Three nutritive enemata were given at intervals of four hours. The first one was not retained. Half hour before the second enema a suppository of tannic acid was given, and the remaining two were retained.

April 22d, 7.30 P.M.—Temp., right axilla, 100. Temp., left axilla, 97¾.

9.30 P.M.—Temp., right axilla, 100. Temp., left axilla, 95.

April 23d.—Patient about the same; recognized friends who called. Bowels constipated; retention of urine. Pulse 80, resp. 22.

Fed by enemata and stomach pump.

April 24th.—No change. Patient conscious.

" 25th.—Patient perspires on right side of face alone, and right arm. Conjugate deviation of eyes to left. Rigidity still present in right leg, but not in arm.

April 26th.—Pulse 75, resp. 24.

" 27th.—Patient smiles at remarks made, and sometimes cries. Still has aphemia; vomits occasionally.

April 28th.—No change.

" 29th.—Can move right leg a little.

" 30th.—Patient shows more intelligence, tries to speak. Swallows a little for first time.

May 1st.—Continues to swallow very well. Bowels opened with ox-gall enemas. Is more intelligent.

	RIGHT AXILLA.	LEFT AXILLA.
May 3d, 8 A.M.—	Temp. 99	Temp. 99
7.30 P.M.	" 98½	" 98
" 4th, A.M.	" 99	" 99
P.M.	" 99½	" 98
" 5th, A.M.	" 99½	" 96
P.M.	" 99	" 98½
" 6th, A.M.	" 99¼	" 95½
P.M.	" 99½	" 97
" 7th, A.M.	" 99	" 98
P.M.	" 100	" 99

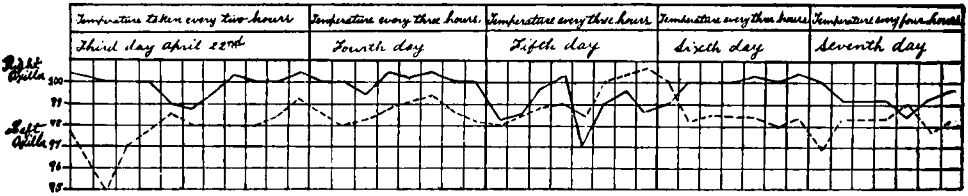
Pulse 80, resp. 18. Patient has pain in the right arm and leg, which she tries to move with her left hand.

	RIGHT AXILLA.	LEFT AXILLA.
May 7th, 12 P.M.—	Temp. 100	Temp. 98½
" 8th, A.M.	" 99	" 96
P.M.	" 99	" 96¼
12 P.M.	" 99½	" 96
" 9th, A.M.	" 99	" 98
P.M.	" 99	" 98¼

Patient still complains, by means of signs, of pain in her right arm and leg. She usually retains her food well, but vomited once.

May 10th.—Patient shows signs of more intelligence. She moves her right leg rather freely and her right arm a little. She has not yet spoken.

		RIGHT AXILLA.	LEFT AXILLA.
May 10th,	A.M.	—Temp. 98	Temp. 98½
	P.M.	" 99½	" 98
" 11th,	A.M.	" 98	" 98½
	P.M.	"	"
" 12th,	A.M.	" 98	" 98½
	P.M.	" 99½	" 99
" 13th,	A.M.	" 97	
	P.M.	" 99½	
" 14th,	A.M.	" 98	
	P.M.	" 99	
" 15th,		" 100	
" 16th,		" 99	



Patient keeps her right leg flexed all the time, unless it be extended by force. She moves it much more freely than her right arm. The leg is carefully but fully extended morning and evening. Patient eats with evident relish, and retains eggs, corn-starch, milk, beef-tea, etc. Bowels regular. Urine passed naturally.

May 17th.—Reflex less. The contracted muscles do not relax in sleep.

May 22d—Extension apparatus with 4½ lb. weight attached to contracted right leg.

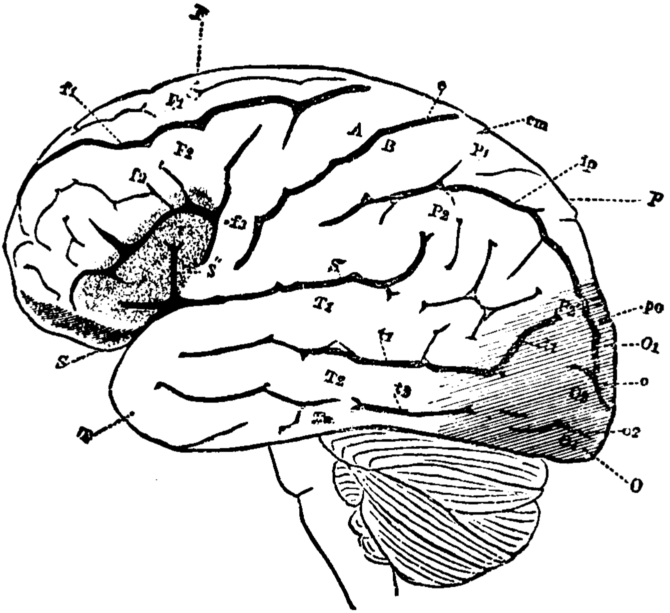
May 24th.—Vomited three times.

May 31st.—Temperature no longer taken. Extension still used. No vomiting. Bowels regular.

During June patient gradually became weaker. *Sensation continued absolutely abolished in the right half of the face* *and imperfect in the arm and leg. The left lower extremity continues to show rigidity, Vomiting occurs daily. Bed-sores developed on nates and sacrum.*

On June 16th she died.

Autopsy held twelve hours after death. Body somewhat emaciated.



Case I.—Thrombosis of branch of left middle cerebral.

Brain very œdematous, its tissue not firm. Over the 2d and 3d left frontal convolutions was seen a light brownish discoloration, somewhat depressed and soft. The left occipital convolution seemed softer to the touch than the middle. The arteries at the base were very atheromatous and patent. The cerebral arteries were followed up and no occlusion found. In the 3d left frontal convolution, extending into 2d and slightly into precentral, and into the island of Reil, was a yellow softening. The tissue was yellowish, of nearly the consistence of pus, which it resembled in gross appearance, but under the microscope showed only granular and fatty matter with many compound granular corpuscles. The softened space was of the size of a pigeon's egg, and was just in front and outside of the caudate nucleus, the outer portion of which was slightly involved. The cortex over the affected part was profoundly affected. Nearly the whole of left occipital lobe and part of the parietal were white and soft, but not broken at all, evidently a recent change. There was no evidence of a hæmorrhage there. Most of the parietal lobe, the basal ganglia, and the right side of the brain were normal; also the cerebellum, pons, and medulla. Ventricles normal.

Heart.—Mitral valves very slightly thickened, and two to three patches of atheroma on endocardium.

Other organs normal.

Probable blocking of the 1st (and 2d) branches of the middle cerebral.

The points of interest here are the unilateral fever and hyperidrosis, dysphagia, motor aphasia, early rigidity without involvement of the ventricles, total anæsthesia of the face.

The occipital lesion was evidently a late one, and could not have had any relation with the anæsthesia which was present from the onset.

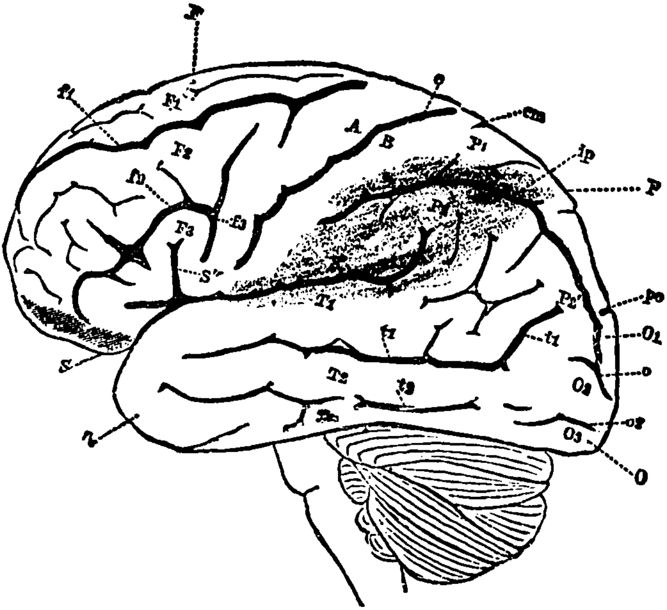
CASE II.—A. D., female, age 60.

The history of this case is very imperfect, as the patient when seen was entirely aphasic. I depend on the statement of friends, who assured me that five years before she had had a total left-sided paralysis, and that for some time she had no feeling in the right leg. From the positive statements made, and the fact that the symptom was remembered so long, it must be concluded that the sensory disturbance was very great.

She was brought to the hospital, February 22d, with right-sided hemiplegia and complete motor aphasia; no anæsthesia noted, intelligence being preserved. She died next day.

Autopsy: The brain showed a recent hæmorrhage in the left hemisphere and an extensive convolutional atrophy in the right hemisphere due to a lesion which must have involved the supramarginal gyrus, part of superior parietal, and part of middle third of posterior central, and part of first temporal.

There was a blocking up probably of the third branch of the Sylvian artery.



CASE II.—Right hemisphere.

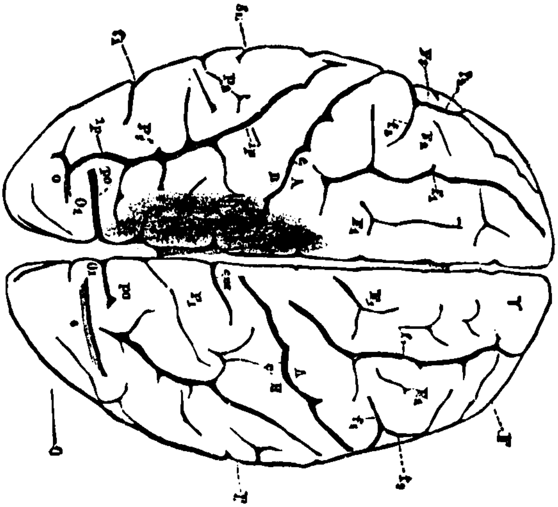
CASE III.—The specimens in this case were shown, and the case reported briefly at a meeting of the N. Y. Neurological Society (*Medical Record*, March 5th, 1887, p. 280).

Pachymeningitis hæmorrhagica, with large meningeal hæmorrhage pressing chiefly on leg-centre—right hemiplegia, total paralysis in leg, aphasia, hemianæsthesia, convulsions limited to arm and face.

The patient was a woman about 68 years old, and came into the hospital with complete motor aphasia, and unable to give any previous history. She had no paralysis at first, but three days after admission she had a general convulsion, followed by right hemiplegia—total in the leg, and some right-sided analgesia. On the second and third days she had a series of brief localized convulsions, involving the face bilaterally and the right arm. The movements were clonic, beginning in the muscles of the lower jaw. The other peculiarities were these:

1. The pupils remained small during the convulsions.

2. The conjugate deviation of the eyes was at first, and very temporarily, toward the side of the lesion, and away from the paralyzed side. The head also was turned toward this side. When this occurs it is ordinarily spoken of as a paralytic deviation. This does not explain it here, since almost immediately the head and eyes were turned strongly to the opposite and paralyzed side.



CASE III.—The darkened area indicates where the severe compression was found.

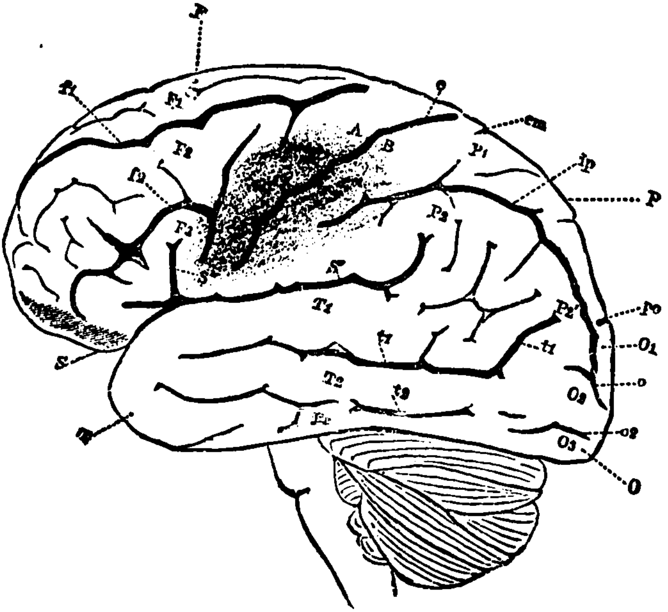
3. The temperature on the paralyzed side was one degree higher than normal, and higher by a degree than that of the other side. In meningeal hæmorrhages the temperature is often below normal, according to Minot.

4. The presence of hemianæsthesia.

The patient died on the third day. Post-mortem showed chronic pachymeningitis over both convexities, but more on the left side. On the left convexity there was a very extensive, fresh meningeal clot, pressing upon and flattening especially the upper half of the central convolutions and the superior parietal lobule. Brain substance normal.

CASE IV.—This case, taken from the Bellevue Hospital records, though imperfectly recorded, seems to be of some value.

A woman, aged 58, was admitted to the hospital with a history of having had a sudden attack of right hemiplegia. On admission she was entirely aphasic, but conscious. The face was but little paralyzed, but there was a total loss of motion and sensation in the right arm and leg.



CASE IV.

Autopsy showed a thrombosis of a branch of the left middle cerebral, involving the middle and part of the lower third of both central convolutions, and the white substance beneath, and the anterior part of the corpus striatum (caudate nucleus). The internal capsule was normal.

There was a probable blocking of the second branch of the Sylvian artery and perhaps of anterior central artery.

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The cases are arranged as numbered in the table. The first forty-seven cases are taken from Luciani and Sepelli's work, and are numbered in the order as given by those authors. These cases are given in abstract, and I have not gone over the original reports myself. They are analyzed in my tables, however.

Thirty-three cases are taken from the table prepared by Starr (*Jour. Nerv. and Men. Disease*, July, 1884, p. 401). All of these, as well as the other cases reported by that writer, I have verified. The remaining sixty-four cases are those collected by myself, including the three original cases reported by myself.

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DISCUSSION ON DR. DANA'S PAPER.

DR. C. K. MILLS considered the paper interesting and valuable, yet its inferences were not, he thought, sustained. He had himself recently gone over a large number of cases with the same object as Dr. Dana. It was probable that the cases gone over by both were at least in part the same. He had concluded that in the limbic lobe, the precuneus, and the postero-parietal region were probably situated the cortical areas for cutaneous sensation. In more than thirty out of the forty-one cases collected by Dr. Starr, the lesions involved the superior or inferior parietal lobule in addition to the central convolutions. Dr. Starr had quoted four of his own cases. In all of these he thought the lesion sufficiently deep to have involved the tracts going to or coming from the gyrus fornicatus, the procuneus, or the postero-parietal region. Lesions of association fibres between related sensory and motor areas would, he thought, account for some of the sensori-motor phenomena. The speaker referred to a case in which the fibroma, starting from the first and second frontal convolution, invaded the gyrus fornicatus and the corpus callosum. It did not involve the optic or trigeminal nerves, yet conjuntival anæsthesia and ulcerative keratitis were present. In a case of Loffler, referred to by Hitzig, where the skull was driven in about median line in the leg region, both legs were hyperæsthetic, probably from actual or inflammatory invasion of the gyrus fornicatus; Dr. Mills spoke of several cases of his own. To say the least, the question was still an open one ; for himself he believed that cortical sensory centres were distinct from cortical motor centres.

DR. M. A. STARR considered Dr. Dana's paper one of great value. All admitted that the location of the cortical sensory centers was an open question, hence very addition to to the facts was of exceeding value. Dr. Mills, in his remark that it was impossible to tell whether fibres were sensory or motor, did not recognize the fact that the most of the work done in tracing fibres was now done upon the brains of fœtuses where the distinction between the sensory and motor tracts could be readily made out by the time and direction of their developement. In the posterior part of the internal capsule, behind the pyramidal tract there was a tract known to be sensory, going to the parietal and the post central convolutions. The direction of this bundle of fibres was outward, not into the gyrus fornicatus or to the limbic lobe. These facts aided the view advanced by Dr. Dana, and not the view of Ferrier advanced by Dr. Mills.

In comparative anatomy, Zuckerkandl had on the contary connected the sense of smell with the gyrus fornicatus, which was small in animal, having but a slight sense of smell, though they might have a large general sensory surface ; and conversely large, where the sense of smell was pronounced. While many of the cases in his own collection had been unsatisfactory, there had been none among them which would support the theory of Ferrier, that the centre or general sensation is in the gyrus fornicatus. As to the case which Dr. Mills described as a lesion invading the hippocampus, the crus cerebri was so near that the invasion of the hippocampus without the involvement of the crus would be most difficult of determination. We have undoubtedly a sensory tract in the crus, presumably in the lemniscus. Ferrier in his last edition could mention only one instance in which in experiment upon the hippocampus the crus was not injured. All of these first experiments were performed too, before the adoption of antisepsis and for that reason are unreliable. As to the other instance mentioned by Dr. Mills, it was difficult to think that the trigeminal nerve was not involved, and the probability seemed in favor of a basilar affection involving it.

The signal symptoms of cortical epilepsy, numbness and tingling, showed that there must be a connection between the cortical motor regions and those of sensation, and the fact that the sensory disturbance usually preceded the motor, contraindicated the view that the sensory areas were distant from the motor.

Another fact had to be taken into consideration in interpreting lesions of the cortex. Each half of the body was represented in both sides of the brain so that lesion of one side of the brain would cause only partial anæsthesia with compensation by the other half for the action lost.

Dr. SEGUIN stated that the results of extirpation of the motor zone would support Schiff's theory of a sensori motor function. The peculiar attitude of the fore paw in Schiff's dogs which was dragged without actual paralysis was explainable only by a degree of anesthesia of the foot. The aura of localized epilepsy (which is often a sensory signal symptom) in brain tumor was another evidence in its favor. The speaker referred to a case of non-syphilitic, brain tumor cured by large doses of the potassium iodide. In this case the convulsion commenced in the hand of the left side and extended to the forearm, arm, face and leg; then, extended to the other side when consciousness was lost. These attacks always commenced with the sensation of wires "working" in forearm and hand. The attacks came at longer and longer intervals and were more restricted until only the arm was affected, and finally the attacks ceased. Long after the patient denied any *visible* attack, however, the sensation of wires boring into the hand would recur at long intervals.

Another argument was furnished by the results o operation. The speaker's case upon which Dr. Weir had operated supported the conclusions of Dr. Dana. Before the operation the man had had some anesthesia of the right hand and cheek. Since the operation this had been increased. It was not complete but was still very marked.

It was most marked for tactile sensation ; analgesia was slight, and the muscular sense normal. The anesthesia affected the right hand, cheek, the right half of the tongue and the inside of the mouth. The tumor removed was deeply situated and involving the caudal extremity of the second frontal and the edge of the precentral convolution. A space about the size of a silver dollar was excised. There was no lesion of the fundus, no petit mal. In addition to the anesthesia there was paresis of the arm and cheek.[1]

DR. DANA acknowledged that with a special sense such as the tactile sense, it seemed more rational that there should be a special centre. He had started in his study of the subject strongly prejudiced in favor of the gyrus fornicatus. The facts of pathology were however opposed to the existence of a sensory centre in the limbic lobe. The speaker referred to the case of Exner in which the anterior half of the limbic lobe was softened without sensory symptoms. On the other hand the lesions of the cortex which gave rise to anæsthesia were not always deep. Luciani and Sepelli had reported several cases of simple superficial softening of the cortex in which anesthesia was present.

In regard to Dr. Starr's suggestion, he thought that it could not be borne out. The sensory fibres crossed in the cord, and almost complete anesthesia was produced by section on the internal capsule, medulla, or cord.

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[1] Cf. American Journal of the Medical Sciences, July, Aug. and Sept. 1888.